PAROXYSMAL VENTRICULAR TACHYCARDIA OCCURRING IN A NORMAL HEART

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It is widely taught—and rightly so—that paroxysmal ventricular tachycardia is one of the rarer arrhythmias, and is associated with grave myocardial damage, with special reference to myocardial infarction. It is the least common, and most serious of the paroxysmal tachycardias, which contain four varieties of arrhythmia: supra-ventricular (auricular and nodal) 60 per cent.; auricular fibrillation, 30 per cent.; auricular flutter, 6 per cent.; and ventricular tachycardia, 4 per cent.

Figures from various sources (Campbell, 1947) would indicate that in the latter group, four-fifths of the cases had seriously damaged hearts. In the remaining fifth no cause was found for the paroxysms. Paroxysmal ventricular tachycardia is more common in men than in women in the proportion of about 3.2. This arrhythmia was first identified by Sir Thomas Lewis in 1909 and Gallavardin (1920, 1921, 1922) emphasized the seriousness of the 'terminal pre-fibrillatory ventricular tachycardia,' and also described a less common type characterized by short attacks with frequent extrasystoles. Also at this time work was published by various authors which stressed myocardial infarction as the precipitating cause of the attacks, and pointed out the therapeutic action of quinidine in these. Froment (1932) classified paroxysmal ventricular tachycardia and included a subgroup of paroxysms occurring in normal hearts, and since then several large series of cases have been reported, among which were Campbell (1947) and Armbrust and Levine (1950).

Aids to the diagnosis of paroxysmal ventricular tachycardia by the bedside are said to be varied intensity of the first heart sound and slight irregularity on careful auscultation; although these may be present, their appreciation by the human ear at rates of 200 per minute is another matter. Campbell considers that the rate per minute does not help in distinguishing ventricular and auricular tachycardias, and that the faster rates were found in the more healthy hearts because these alone could survive at these rates. Vagal stimulation

by sinus pressure will, of course, not affect the rate in ventricular tachycardia.

In 1953 Froment, Gallavardin and Cahen offered a classification of various forms of paroxysmal ventricular tachycardia, and included some case report. They described the following groups:—

- (i) Terminal prefibrillatory ventricular tachycardia.
- (ii) Curable and mild monomorphic extrasystoles, with paroxysms of tachycardia.
- (iii) Paroxysmal ventricular tachycardia due to a lesion of the ventricular septum.
- (iv) Persistent and prolonged ventricular tachycardia developing in sound hearts, usually in young subjects.

Case Report

A married woman, aged 48, first became aware in 1948 of a paroxysm which caused alarm, faintness and collapse. It lasted only a short time, and past history was completely negative from the point of view of cardiac symptoms, rheumatic phenomena, etc. At this stage she was seen at the National Heart Hospital, and clinical, radiological and electrocardiographic examination was normal. Later she became aware of a 'fluttering in the throat' from time to time, which gradually seemed to pass off after a few hours, and give rise to no real discomfort once she became used to it.

In October 1953 the paroxysms became more prolonged and she sought medical advice. She was seen in St. James's Hospital, Balham. At that time her E.C.G. was normal, and no abnormality was found on clinical examination except occasional extrasystoles. She continued to complain of attacks, and quinidine was employed, but this merely added nausea and sweating to the discomfort of the paroxysms which were then lasting up to days at a time.

In November 1953 the first E.C.G. of the arrhythmia was obtained which illustrated numerous extrasystoles occurring at a rate of about 200

Showing paroxysms of monomorphic ventricular extrasystoles interspersed with normal sinus beats (8.12.1953).

per minute, interspersed with normal sinus beats every 10-15 seconds. Sinus pressure was ineffective. The patient experienced a vague fluttering sensation in the neck, but never angina or dyspnoea.

Quinidine was discontinued, and she was given pronestyl (procaine amide), 250 mg. t.d.s. This appeared to revert her to normal rhythm, but after 24 hours the same type of paroxysm recurred. An increase in the dose of pronestyl again appeared to revert her to normal rhythm, but when seen later in the out-patients' department it was evident that pronestyl in doses of up to 3 g. daily was having no effect on the arrhythmia.

The patient thinks she can initiate a paroxysm by stretching her arms above her head, or making a sudden movement. The commencement of a paroxysm causes no discomfort, but if it lasts for 24 hours or more causes her to feel faint and uncomfortable. When in normal rhythm no abnormality was noted apart from an occasional extrasystole. There is no evidence of coronary ischaemia; chest X-ray was normal; full blood count was normal; B.M.R. was minus 0.5; and W.R. and Kahn were negative.

A trial withdrawal of pronestyl in February 1954 seemed to increase the severity and duration of the paroxysms, so that the patient took to bed for a few days, and the drug has now been resumed in doses of 3 g. daily. When last seen in March 1954 no abnormality was found, and although she is having paroxysms lasting hours every other day, is able to carry on her work as a shop assistant.

Froment, Gallavardin and Cahen quote two case reports of this type, who were resistant to treatment, and who were followed from the ages of 33-64 (female) and 17-41 (male) when the paroxysms gradually ceased.

Discussion

This disorder is characterized by an almost

permanent extrasystolic irregularity, interspersed with paroxysms of tachycardia of brief duration. In more severe forms these become longer, until they coalesce into prolonged attacks which are interrupted by sinus beats only at infrequent intervals. It is found in young subjects with healthy hearts, and is usually very resistant to therapy, including quinidine. The paroxysms are strictly monomorphic, and the isolated extrasystoles have the same contours as those constituting the paroxysms.

Very rarely episodes of transient heart failure can occur when the episodes are severe and prolonged, and occasionally faintness or vertigo may occur at the beginning of an attack. In two cases described by Froment, Gallavardin and Cahen the ectopic rhythm disappeared, and after many years there were no abnormal findings on examination. The authors suggest that the lesion may be a microscopic myocardial injury, probably of the bundle of His acting as an 'epine irritative.'

This type differs from Type IV which is characterized by prolonged ventricular paroxysms without a break—analogous clinically to supra-ventricular paroxysmal tachycardia. Clinically the beginning and end of the paroxysms are abrupt; these often last some days, and are well tolerated. They do not conform to Type II as there are neither isolated extrasystoles nor any short periods of normal beats.

Post-paroxysmal E.C.G. changes are often seen in Type IV involving inversion of the T waves but these are apparently of no prognostic significance. Quinidine was successful in terminating these paroxysms in contrast to those of Type II which were resistant to treatment.

In the case report of Type III the paroxysms ceased after successful W.R. conversion, and syphilitic septal lesion was assumed.

Summary

A case of idiopathic paroxysmal ventricular tachycardia is reported. Some of the literature on paroxysmal ventricular tachycardia is reviewed, including a classification of types of this arrhythmia.

The characteristic features of the two main types occurring in apparently normal hearts is discussed, including the resistance to treatment of the type characterized by extrasystoles, with paroxysms of tachycardia.

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